QUIZ NAVIGATION

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Grade	**5.00** out of 10.00 (**50**%)

Question 1

ID: 38888

Correct

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You are a pharmacist working at a local community pharmacy and are approached by one of your patients. ST is a 66-year-old African-American woman who is looking for advice on "kidney protection". A few weeks ago, her sister was diagnosed with acute kidney injury (AKI) and hospitalized for a week. ST would like to know how she can prevent AKI and whether she is at risk for it. You take a look at her chart and obtain the following information:

ST's past medical history is significant for hypertension, hypothyroidism, type 2 diabetes mellitus, and myocardial infarction requiring coronary artery bypass graft (CABG) surgery 3 years ago.

Her current medications include rosuvastatin 40 mg daily, acetylsalicylic acid 81 mg daily, levothyroxine 75 mcg daily, metformin 500 mg BID, perindopril 8 mg daily, and metoprolol 100 mg BID

All of the following options are known risk factors that ST has for developing acute kidney injury (AKI) **EXCEPT**:

Select one:

- African-American race ✖
- History of cardiac surgery ✖
- Hypothyroidism ✔ — *Rose Wang (ID:113212) this answer is correct. Having hypothyroidism is not a known risk factor for developing acute kidney injury (AKI).*
- Diabetes ✖

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To identify known risk factors for developing acute kidney injury (AKI).

BACKGROUND:

The incidence of acute kidney (AKI) in the community setting is very low compared to acute hospital settings. This is because hospitalized patients are more likely to develop AKI as patients are more prone to depleted plasma volumes (due to blood loss following surgeries, dehydration, and/or urinary catheter implantation) in these settings. Development of AKI is associated with increased morbidity; AKI can lead to longer hospitalizations, readmission, ventilator days, and need for post-hospitalization care. In the last 2 decades, the total number of AKI-related hospitalizations has increased 4-fold from 953,926 in 2000 to 3,959,560 in 2014. AKI tends to be more common in the critically ill hospitalized patient population (30-60%) compared to the non-critically ill hospitalized patients (3-18%).

Risk factors that increase risk of AKI include CKD, diabetes, cardiac or hepatic disease, albuminuria, major surgery (especially cardiac surgery), acute decompensated heart failure, sepsis, hypotension, fluid loss (diarrhea, vomiting, or dehydration), medications (angiotensin-converting enzyme [ACE] inhibitors, angiotensin receptor blockers [ARBs], lithium, aminoglycosides, etc.), advanced age, male gender, and African American race. Up to 60% of AKI cases are drug-induced.

70% of AKI cases are pre-renal AKI, which is due to decreased perfusion to the kidneys. They are typically caused by conditions that decrease cardiac output and perfusion (e.g., dehydration, severe blood loss, sepsis, severe burns, and cirrhosis), renal arterial disease, and medications (such as ACE inhibitors, diuretics, and NSAIDs). ACE inhibitors and ARBs inhibit angiotensin-II activity which normally would constrict the efferent arteriole when the perfusion pressure in the kidneys is low. If perfusion is low, filtration remains low and this results in poor kidney function. NSAIDs inhibit the production of prostaglandins which normally promotes vasodilation of the afferent arteriole when perfusion is low. Diuretics promote diuresis which leads to depleted blood volume and decreased perfusion.

Intrinsic renal failure makes up 20% of the AKI cases. Acute tubular necrosis (ATN) can be caused by ischemia (due to hypotension or sepsis), nephrotoxic substances (such as lithium, radiocontrast dye, aminoglycosides, cisplatin, carboplatin, cyclosporine, and tacrolimus). Not only is lithium a nephrotoxic medication, but it leads to desensitization of antidiuretic hormone (ADH). Desensitization of ADH leads to inability to concentrate urine and increased diuresis (this condition is known as diabetes insipidus). Acute interstitial nephritis (AIN) is typically caused by medications such as penicillin and nonsteroidal antiinflammatory drugs (NSAIDs). Glomerulonephritis is caused by glomerular diseases, autoimmune diseases (e.g., systemic lupus erythematosus (SLE)), and medications (e.g., lithium and NSAIDs).

Post renal typically occurs due to obstruction or hypertrophy of renal pelvis, ureters, and/or bladder. Obstructions include nephrolithiasis (kidney stones), nephrocalcinosis (calcium deposits along the urinary tract), and bladder outlet obstruction (improper urinary catheter placement leading to blockage of urinary tract). It can also be caused by malignancies, medications that cause urinary retention (e.g., anticholinergic medications), other medication (e.g., methotrexate, penicillin, and sulfamethoxazole trimethoprim (Septra)).

RATIONALE:

Correct Answer:

(Option #3): Having hypothyroidism is not a known risk factor for developing acute kidney injury (AKI).

Incorrect Answers:

(Option #1): Being of African-American race is a known risk factor for developing acute kidney injury (AKI).
(Option #2): Having a history of major surgery, especially cardiac surgery, is a known risk factor for developing acute kidney injury (AKI).
(Option #4): Having diabetes is a known risk factor for developing acute kidney injury (AKI).

TAKEAWAY/KEY POINTS:

Risk factors that increase risk of AKI include CKD, diabetes, cardiac or hepatic disease, albuminuria, major surgery (especially cardiac surgery), acute decompensated heart failure, sepsis, hypotension, fluid loss (diarrhea, vomiting, or dehydration), medications (angiotensin-converting enzyme [ACE] inhibitors, angiotensin receptor blockers [ARBs], lithium, aminoglycosides, etc.), advanced age, male gender, and African American race.

REFERENCES:

[1] Flurie RW. Disorders of Potassium and Magnesium Homeostasis. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

[2] Kantorvich, A. Medications That Always Use Actual Body Weight to Calculate Creatinine Clearance. *PharmacyTimes*. Published June 3 2016.

[3] Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Workgroup. KDIGO clinical practice guideline for acute kidney injury. *Kidney Int Suppl.* 2012;2:1–138.

[4] Maker J, Roller L, Dager W. Acute Kidney Injury. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

The correct answer is: Hypothyroidism

Question 2

ID: 38889

Correct

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You are a renal pharmacist working at a large teaching hospital. Today, one of your students approaches you for some advice. She is trying to calculate the estimated glomerular filtration rate (eGFR) for one her patients using the Cockcroft-Gault equation as follows:

eGFR = [140 - age (years)] x ideal weight (kg)] / [serum creatinine (mg/dL) x 72]

She has forgotten how to use this equation and would like you to refresh her memory.

All of the following options regarding the Cockcroft-Gault equation to calculate estimated glomerular filtration rate (eGFR) are true **EXCEPT**:

Select one:

- The estimated glomerular filtration rate (eGFR) calculated using the Cockcroft-Gault equation needs to be multiplied by 0.85 for women ✖
- The estimated glomerular filtration rate (eGFR) calculated using the Cockcroft-Gault equation provides a real-time prediction of renal function decline during acute kidney injury (AKI) ✔

Rose Wang (ID:113212) this answer is correct. The estimated glomerular filtration rate (eGFR) calculated using the Cockcroft-Gault equation may not reflect the renal function decline during acute kidney injury (AKI) because serum creatinine level changes lag about 1-2 days after the onset of kidney injury.

- Actual Body Weight (ABW) should be used to calculate the estimated glomerular filtration rate (eGFR) with the Cockcroft-Gault equation if the patient is less than their Ideal Body Weight (IBW) ✖
- Adjusted Body Weight (AdjBW) should be used to calculate the estimated glomerular filtration rate (eGFR) with the Cockcroft-Gault equation if the patient is obese ✖

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To understand how to use the Cockcroft-Gault equation to calculate the estimated glomerular filtration rate (eGFR).

BACKGROUND:

Acute renal failure, also known as acute kidney injury (AKI), is classified as a sudden decrease in kidney function.

More specifically, AKI is defined as any of the following:

1. Increase in serum creatinine (SCr) by 26.5 µmol/L within 48 hours
2. Increase in serum creatinine (SCr) to 1.5 times baseline within the prior 7 days
3. Urine volume <0.5 ml/kg/h for 6 hours

It is typically caused or worsened by medications and/or disease states and is usually reversible. AKI is characterized by a high SCr concentration and/or low urine output (oliguria). Pre-renal AKI is characterized as oliguria (<400 ml/day although some sources say <500 ml/day). Creatinine is a waste product produced by muscles that gets excreted by the kidney. One of the main roles of the kidneys is to filter waste from the plasma and excrete the waste products in the urine. The concentration of serum creatinine can be used to calculate creatinine clearance (CrCl) and the estimated glomerular filtration rate (eGFR) which is used to determine a patient's kidney function. It is important to note that during AKI, eGFR may not reflect the renal function decline because serum creatinine level changes lag about 1-2 days after onset of kidney injury.

Likewise, eGFR calculations may also not be reflective of when kidney function begins to improve. They are best used as a representation of kidney function when serum creatinine is at steady-state. During AKI, percent changes of serum creatinine may be preferred compared to absolute changes. eGFR is calculated using the following equation:

Cockcroft-Gault Equation:

eGFR = [140 - age (years)] x ideal weight (kg)] / [serum creatinine (mg/dL) x 72] x 0.85 (if female)

Note: Weight should be Ideal Body Weight (IBW) unless the patient is obese (if a patient is 30% over their IBW) or if the patient is less than their IBW. If patients weigh less than their IBW, their Actual Body Weight (ABW) should be used instead. Obese patients should use an adjusted body weight (AdjBW) which applies a correction factor of 40% to the IBW.

IBW (males)= 50 kg + (2.3 x every inch over 5 feet)

IBW (females) = 45.5 kg + (2.3 x every inch over 5 feet)

AdjBW= IBW + (0.4 x IBW)

RATIONALE:

Correct Answer:

(Option #2): The estimated glomerular filtration rate (eGFR) calculated using the Cockcroft-Gault equation may not reflect the renal function decline during acute kidney injury (AKI) because serum creatinine level changes lag about 1-2 days after the onset of kidney injury.

Incorrect Answers:

(Option #1): The estimated glomerular filtration rate (eGFR) calculated using the Cockcroft-Gault equation needs to be multiplied by 0.85 for women.
(Option #3): Actual Body Weight (ABW) should be used to calculate the estimated glomerular filtration rate (eGFR) with the Cockcroft-Gault equation if the patient is less than their Ideal Body Weight (IBW).
(Option #4): Adjusted Body Weight (AdjBW) should be used to calculate the estimated glomerular filtration rate (eGFR) with the Cockcroft-Gault equation if the patient is obese, because the AdjBW applies a correction factor of 40% to the ideal body weight (IBW).

TAKEAWAY/KEY POINTS:

The estimated glomerular filtration rate (eGFR) calculated using the Cockcroft-Gault equation may not reflect the renal function decline during acute kidney injury (AKI) because serum creatinine level changes lag about 1-2 days after the onset of kidney injury.

REFERENCE:

[1] Flurie RW. Disorders of Potassium and Magnesium Homeostasis. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

[2] Kantorvich, A. Medications That Always Use Actual Body Weight to Calculate Creatinine Clearance. *PharmacyTimes*. Published June 3 2016.

[3] Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Workgroup. KDIGO clinical practice guideline for acute kidney injury. *Kidney Int Suppl.* 2012;2:1–138.

[4] Maker J, Roller L, Dager W. Acute Kidney Injury. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

The correct answer is: The estimated glomerular filtration rate (eGFR) calculated using the Cockcroft-Gault equation provides a real-time prediction of renal function decline during acute kidney injury (AKI)

Question 3

ID: 38890

Correct

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All of the following options are appropriate goals of therapy for acute kidney injury (AKI) **EXCEPT**:

Select one:

- Cure pre-existing chronic kidney disease (CKD) ✔

Rose Wang (ID:113212) this answer is correct. Curing any pre-existing comorbid chronic kidney disease (CKD) is not an appropriate goal of therapy for acute kidney injury (AKI). Furthermore, there is no cure for CKD.

- Correct and maintain electrolyte, acid-base, and mineral balance ✖
- Minimize secondary organ damage ✖
- Manage effects of decreased renal function ✖

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To identify the goals of therapy for acute kidney injury (AKI).

BACKGROUND:

The goals of therapy for acute kidney injury (AKI) include:

- Preserve and optimize renal function
- Correct and maintain electrolyte, acid-base, and mineral balance
- Minimize secondary organ damage from AKI
- Manage effects of decreased renal function (e.g., hyperkalemia)

RATIONALE:

Correct Answer:

(Option #1): Curing any pre-existing comorbid chronic kidney disease (CKD) is not an appropriate goal of therapy for acute kidney injury (AKI). Furthermore, there is no cure for CKD.

Incorrect Answers:

(Option #2): Correcting and maintaining electrolyte, acid-base, and mineral balance is an appropriate goal of therapy for acute kidney injury (AKI).
(Option #3): Minimizing secondary organ damage is an appropriate goal of therapy for acute kidney injury (AKI).
(Option #4): Managing the effects of decreased renal function (e.g., hyperkalemia) is an appropriate goal of therapy for acute kidney injury (AKI).

TAKEAWAY/KEY POINTS:

The goals of therapy for acute kidney injury (AKI) include preserving and optimizing renal function, correcting and maintaining electrolyte, acid-base, and mineral balance, minimizing secondary organ damage from AKI, and managing effects of decreased renal function (e.g., hyperkalemia).

REFERENCES:

[1] Flurie RW. Disorders of Potassium and Magnesium Homeostasis. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

[2] Kantorvich, A. Medications That Always Use Actual Body Weight to Calculate Creatinine Clearance. *PharmacyTimes*. Published June 3 2016.

[3] Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Workgroup. KDIGO clinical practice guideline for acute kidney injury. *Kidney Int Suppl.* 2012;2:1–138.

[4] Maker J, Roller L, Dager W. Acute Kidney Injury. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

The correct answer is: Cure pre-existing chronic kidney disease (CKD)

Question 4

ID: 38891

Incorrect

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Which of the following options are associated with post-renal acute kidney injury (AKI)?

Select one:

- Rhabdomyolysis ✖ — *Rose Wang (ID:113212) this answer is incorrect. Rhabdomyolysis is associated with intrinsic kidney damage.*
- Benign prostatic hypertrophy (BPH) ✔
- Septic shock ✖
- Hepatorenal syndrome ✖

Incorrect

Marks for this submission: 0.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To identify different causes of acute kidney injury (AKI).

BACKGROUND:

Acute kidney injury (AKI) occurs when there is a rapid decrease in kidney function. AKI can be classified based on where the damage or impairment is occurring in the kidney:

- *Pre-renal AKI* occurs when there is hypoperfusion of the kidney (the most common cause of AKI)
- *Intrinsic AKI* occurs when there is damage or impairment within the kidney
- *Post-renal AKI* occurs when a severe blockage beyond the kidney (e.g., in the ureter) causes waste buildup in the kidney

Possible causes of AKI

Impairment Location	Possible causes of AKI
Pre-renal AKI	• Hypovolemia • Increased vascular resistance • Reduced cardiac function • Systemic vasodilation
Intrinsic AKI	• Bilateral renal artery stenosis • Infection • Immune system dysfunction (e.g., lupus, IgA glomerulonephritis) • Nephrotoxic drugs
	• Blood clots

Post-renal AKI	• Improperly placed catheter • Kidney stones (nephrolithiasis) • Urogenital cancers

AKI is defined as any of the following:

- Increase in SCr by ≥26.5 umol/L within 48 hours, or
- Increase in SCr by ≥1.5 times the baseline within 7 days, or
- Urine volume of <0.5 ml/kg/hour for 6 hours

In addition to reduced or lack of urine output, fluid retention is a very common sign of AKI. Nausea/vomiting can also occur as AKI results in a buildup of waste in the blood.

RATIONALE:

Correct Answer:

(Option #2):

Benign prostatic hypertrophy (BPH) can constrict the urethra severely, which can result in urine build-up and limit renal emptying.
Incorrect Answers:

(Option #1): Rhabdomyolysis is associated with intrinsic kidney damage.
(Option #3): Septic shock is associated with pre-renal acute kidney injury (AKI) due to hypotension.
(Option #4): Hepatorenal syndrome is associated with pre-renal acute kidney injury (AKI) due to insufficient renal perfusion.

TAKEAWAY/KEY POINTS:

The kidneys are very complex organs and acute kidney injury (AKI) can result from dysfunction in any part of the organ.

REFERENCES:

[1] KDIGO clinical practice guidelines for acute kidney injury. *Kidney International Supplements*. 2012;2(1). doi:10.1038/kisup.2012.1.

[2] Makris K, Spanou L. Acute Kidney Injury: Definition, Pathophysiology and Clinical Phenotypes. *Clin Biochem Rev*. 2016;37(2):85-98.

The correct answer is: Benign prostatic hypertrophy (BPH)

Question 5

ID: 38892

Incorrect

Flag question

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THE NEXT TWO QUESTIONS INCLUSIVE REFER TO THE FOLLOWING CASE:

VT is an 84-year-old female who resides in a retirement home with her husband. She was brought to the hospital early this morning by ambulance after she suffered a fall. Upon X-ray, it is revealed that VT has a pelvic fracture. The nurse from the retirement home reports that VT has been experiencing vomiting and diarrhea over the past two days. VT's husband reports that late last night, VT was hallucinating that there was a cat in the corner of their room and attempted to catch it, causing her to fall. She has never had a prior history of hallucinations in the past. Her past medical history is significant for type 2 diabetes, hypertension, dyslipidemia, osteoporosis, and hypothyroidism. Her current medications include metformin 500 mg BID, gliclazide extended-release 60 mg once daily, canagliflozin 300 mg once daily, perindopril 8 mg once daily, atorvastatin 20 mg once daily, denosumab 60 mg subcutaneously every 6 months, and levothyroxine 100 mcg once daily. At the hospital, her serum creatinine is revealed to be 122 umol/L, which is noticeably higher than her previous reading of 82 umol/L taken 48 hours ago. The internal medicine physician diagnoses VT with acute kidney injury (AKI) secondary to hypovolemia due to a possible infection and admits her to the hospital as an inpatient.

All of the following options are appropriate non-pharmacological treatment options for VT's acute kidney injury (AKI) **EXCEPT**:

Select one:

- Renal replacement therapy (RRT) ✔
- Temporarily hold medications for sick day management ✖
- Electronic alert system for acute kidney injury (AKI) detection ✖
- Oral caloric intake of 20-30 kcal/kg/day ✖

Rose Wang (ID:113212) this answer is incorrect. The KDIGO guidelines currently recommend caloric intake of 20-30 kcal/kg/day regardless of stage of kidney impairment and oral administration is preferred.

Incorrect

Marks for this submission: 0.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To identify non-pharmacological treatment options for acute kidney injury (AKI).

BACKGROUND:

Non-pharmacological interventions for acute kidney injury (AKI) include implementation of electronic alert systems and nutrition maintenance. The KDIGO guidelines currently recommend caloric intake of 20-30 kcal/kg/day regardless of stage of kidney impairment and oral administration is preferred. Electronic alert systems implemented in an acute care setting can allow for early detection of AKI and monitoring of patients on nephrotoxic drugs. It can allow for faster intervention response and decreased loss of kidney function. However, electronic alerts sometimes get missed due to alert fatigue, the phenomenon related to desensitization of safety alerts due to the vast number of clinically insignificant alerts. A solution to combat alert fatigue is to specifically check alerts for those patients who are already deemed at risk of AKI.

RATIONALE:

Correct Answer:

(Option #1): Renal replacement therapy (RRT) may be considered in certain patients with severe acute kidney injury (AKI), such as those with metabolic acidosis, severe hyperkalemia and/or hypermagnesemia, or fluid overload (especially if the patient has pulmonary edema unresponsive to diuretics). There is not enough information currently to decide if VT requires RRT at this time.

Incorrect Answers:

(Option #2): Sick day management involves stopping certain medications (SADMANS) because these medications rely on adequate kidney function and continuing them could increase a patient's risk of adverse events.
(Option #3): Electronic alert systems implemented in an acute care setting can allow for early detection of acute kidney injury (AKI) and monitoring of patients on nephrotoxic drugs. It can allow for faster intervention response and decreased loss of kidney function.
(Option #4): The KDIGO guidelines currently recommend caloric intake of 20-30 kcal/kg/day regardless of stage of kidney impairment and oral administration is preferred.

TAKEAWAY/KEY POINTS:

Renal replacement therapy (RRT) may be considered in certain patients with severe acute kidney injury (AKI), such as those with metabolic acidosis, severe hyperkalemia and/or hypermagnesemia, or fluid overload (especially if the patient has pulmonary edema unresponsive to diuretics).

REFERENCES:

[1] Flurie RW. Disorders of Potassium and Magnesium Homeostasis. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

[2] Kantorvich, A. Medications That Always Use Actual Body Weight to Calculate Creatinine Clearance. *PharmacyTimes*. Published June 3 2016.

[3] Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Workgroup. KDIGO clinical practice guideline for acute kidney injury. *Kidney Int Suppl.* 2012;2:1–138.

[4] Maker J, Roller L, Dager W. Acute Kidney Injury. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

The correct answer is: Renal replacement therapy (RRT)

Question 6

ID: 38893

Correct

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The internal medicine physician decides to temporarily hold some of VT's medications for sick day management until her clinical condition improves.

All of the following medications should be temporarily held for VT for sick day management **EXCEPT**:

Select one:

- Metformin ✖
- Gliclazide ✖
- Canagliflozin ✖
- Atorvastatin ✔

Rose Wang (ID:113212) this answer is correct. Sick day management involves stopping certain medications (SADMANS) because these medications rely on adequate kidney function and continuing them could increase a patient's risk of adverse events. These medications include sulfonylureas, ACE inhibitors, diuretics, metformin, ARBs, NSAIDs, and sodium glucose co-transporter inhibitors. Statins do not belong to this list.

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To identify medications that should be temporarily held for sick day management.

BACKGROUND:

Patients who have unstable kidney function, are unable to properly take in fluids, and/or are dehydrated, may be advised to follow sick day management principles. Sick day management involves stopping certain medications (SADMANS) because these medications rely on adequate kidney function and continuing them could increase a patient's risk of adverse events. The following medications include: sulfonylureas, ACE inhibitors, diuretics, metformin, ARBs, NSAIDs, and sodium glucose co-transporter inhibitors. In addition, patients who are on insulin require regular blood glucose monitoring and need to adjust their insulin accordingly.

RATIONALE:

Correct Answer:

(Option #4): Sick day management involves stopping certain medications (SADMANS) because these medications rely on adequate kidney function and continuing them could increase a patient's risk of adverse

events. These medications include sulfonylureas, ACE inhibitors, diuretics, metformin, ARBs, NSAIDs, and sodium glucose co-transporter inhibitors. Statins do not belong to this list.

Incorrect Answers:

(Option #1): Sick day management involves stopping certain medications (SADMANS) because these medications rely on adequate kidney function and continuing them could increase a patient's risk of adverse events. These medications include sulfonylureas, ACE inhibitors, diuretics, metformin, ARBs, NSAIDs, and sodium glucose co-transporter inhibitors.
(Option #2): Sick day management involves stopping certain medications (SADMANS) because these medications rely on adequate kidney function and continuing them could increase a patient's risk of adverse events. These medications include sulfonylureas, ACE inhibitors, diuretics, metformin, ARBs, NSAIDs, and sodium glucose co-transporter inhibitors.
(Option #3): Sick day management involves stopping certain medications (SADMANS) because these medications rely on adequate kidney function and continuing them could increase a patient's risk of adverse events. These medications include sulfonylureas, ACE inhibitors, diuretics, metformin, ARBs, NSAIDs, and sodium glucose co-transporter inhibitors.

TAKEAWAY/KEY POINTS:

Sick day management involves stopping certain medications (SADMANS) because these medications rely on adequate kidney function and continuing them could increase a patient's risk of adverse events. These medications include sulfonylureas, ACE inhibitors, diuretics, metformin, ARBs, NSAIDs, and sodium glucose co-transporter inhibitors.

REFERENCES:

[1] Flurie RW. Disorders of Potassium and Magnesium Homeostasis. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

[2] Kantorvich, A. Medications That Always Use Actual Body Weight to Calculate Creatinine Clearance. *PharmacyTimes*. Published June 3 2016.

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[4] Maker J, Roller L, Dager W. Acute Kidney Injury. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

The correct answer is: Atorvastatin

Question 7

ID: 38896

Incorrect

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Which of the following options is **FALSE** about the management of hyperkalemia?

Select one:

- Sodium polystyrene sulfonate can be used as a diuretic for the treatment of hyperkalemia ✔
- Intravenous calcium gluconate can be used to stabilize electrocardiogram readings caused by hyperkalemia ✖
- Intravenous insulin should be used in combination with glucose for the treatment of hyperkalemia ✖
- Nebulized salbutamol can be used for the treatment for hyperkalemia ✖

Rose Wang (ID:113212) this answer is incorrect. Nebulized beta-agonist (i.e., salbutamol) can be used to push potassium into cells and lower serum potassium levels.

Incorrect

Marks for this submission: 0.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To identify treatment options for hyperkalemia as it can be a complication of renal dysfunction.

BACKGROUND:

During acute kidney injury (AKI), potassium levels can elevate due to decreased potassium excretion. As a result, patients may develop hyperkalemia (when K+ >5 mmol/L). Hyperkalemia can cause symptoms varying in severity from mild symptoms (e.g., muscle contractions and fatigue) to severe and life threatening (e.g., cardiac arrhythmias and death). It is critical to monitor and manage potassium levels in patients with AKI. When hyperkalemia is <6 mmol/L it is considered non-life threatening. It can typically be treated with restriction of foods and drinks with potassium (e.g., bananas, orange juice) and discontinuation of drugs that increase potassium levels (e.g., potassium supplements, ACE inhibitors/ARBs, aldosterone antagonists such as spironolactone). If required, consider using IV or oral furosemide or sodium polystyrene sulfonate (Kayexalate®). Kayexalate® is a potassium binder that exchanges potassium for sodium in the gastrointestinal tract to be excreted. Kayexalate® is available as an oral dose or as a rectal enema (if oral cannot be tolerated). Avoid suspending the enema in sorbitol because it increases the risk of intestinal necrosis. Rectal use is contraindicated in patients with bowel dysfunction. Patients' potassium levels should continually be monitored.

In life-threatening hyperkalemia, a more aggressive treatment regimen should be undertaken. Life-threatening hyperkalemia includes the following patients: symptomatic hyperkalemic patients (visible electrocardiogram (ECG) changes), cardiac arrhythmic patients, patients with generalized muscle weakness or paralysis, serum potassium >6 mmol/L, and patients with rapidly increasing potassium levels. First, use IV calcium gluconate to stabilize ECG readings. Next, use IV insulin and beta-agonist (e.g., nebulized salbutamol) to push potassium into cells which lowers serum potassium. Finally, use furosemide or Kayexalate® to excrete potassium from the body.

RATIONALE:

Correct Answer:

(Option #1): Sodium polystyrene sulfonate can be used for the treatment of hyperkalemia; however, it works as a potassium binder and not as a diuretic.

Incorrect Answers:

(Option #2): Intravenous calcium gluconate can be used in patients with life-threatening hyperkalemia as an initial agent to stabilize electrocardiogram readings.
(Option #3): Intravenous insulin can be used to push potassium into cells and lower serum potassium levels, but it should be used in combination with glucose to avoid hypoglycemia.
(Option #4): Nebulized beta-agonist (i.e., salbutamol) can be used to push potassium into cells and lower serum potassium levels.

TAKEAWAY/KEY POINTS:

Sodium polystyrene sulfonate (Kayexalate®) is a potassium binder that exchanges potassium for sodium in the gastrointestinal tract to be excreted. Kayexalate® is available as an oral dose or as a rectal enema (if oral cannot be tolerated). Avoid suspending the enema in sorbitol because it increases the risk of intestinal necrosis. Rectal use is contraindicated in patients with bowel dysfunction.

REFERENCE:

[1] Flurie RW. Disorders of Potassium and Magnesium Homeostasis. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

[2] Kantorvich, A. Medications That Always Use Actual Body Weight to Calculate Creatinine Clearance. *PharmacyTimes*. Published June 3 2016.

[3] Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Workgroup. KDIGO clinical practice guideline for acute kidney injury. *Kidney Int Suppl.* 2012;2:1–138.

[4] Maker J, Roller L, Dager W. Acute Kidney Injury. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

The correct answer is: Sodium polystyrene sulfonate can be used as a diuretic for the treatment of hyperkalemia

Question 8

ID: 38897

Incorrect

Flag question

Send Feedback

You are a pharmacist working at a local community hospital in the acute ambulatory care unit (AACU). FB is a 45-year-old male who presents to the hospital's emergency room. The emergency department physician assesses FB and decides to admit him to the AACU for further testing. The physician suspects acute kidney injury (AKI) and orders a urinalysis, the results for which come back as positive for hematuria.

All of the following options may cause hematuria **EXCEPT:**

Select one:

- Interstitial nephritis ✖ — *Rose Wang (ID:113212) this answer is incorrect. Interstitial nephritis can cause hematuria as it is inflammation of the kidney lining.*
- Kidney tumour ✖
- Hepatorenal syndrome ✔
- Kidney stones ✖

Incorrect

Marks for this submission: 0.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To identify the signs and symptoms of acute kidney injury (AKI).

BACKGROUND:

Acute kidney injury (AKI) occurs when there is a rapid decrease in kidney function. AKI can be classified based on where the damage or impairment is occurring in the kidney:

- *Pre-renal AKI* occurs when there is hypoperfusion of the kidney (the most common cause of AKI)
- *Intrinsic AKI* occurs when there is damage or impairment within the kidney
- *Post-renal AKI* occurs when a severe blockage beyond the kidney (e.g., in the ureter) causes waste buildup in the kidney

Possible causes of AKI

Impairment Location	Possible causes of AKI
Pre-renal AKI	• Hypovolemia • Increased vascular resistance • Reduced cardiac function • Systemic vasodilation
	• Bilateral renal artery stenosis • Infection

Intrinsic AKI	• Immune system dysfunction (e.g., lupus, IgA glomerulonephritis) • Nephrotoxic drugs
Post-renal AKI	• Blood clots • Improperly placed catheter • Kidney stones (nephrolithiasis) • Urogenital cancers

In addition to reduced or lack of urine output, fluid retention is a very common sign of AKI. Nausea/vomiting can also occur as AKI results in a buildup of waste in the blood.

Hematuria is not a sign of AKI, but many of the causes of AKI are associated with hematuria. Therefore, hematuria can be used as a warning that kidney damage is occurring and the person is at risk of AKI if hematuria cause is left untreated.

RATIONALE:

Correct Answer:

(Option #3): Hepatorenal syndrome causes hypoperfusion to the kidney but will not cause hematuria.

Incorrect Answers:

(Option #1): Interstitial nephritis can cause hematuria as it is inflammation of the kidney lining.
(Option #2): Kidney tumours can cause hematuria.
(Option #4): Kidney stones can cause hematuria.

TAKEAWAY/KEY POINTS:

There are warning signs, such as hematuria, that a patient is at risk of acute kidney injury (AKI) if certain conditions are left untreated.

REFERENCES:

[1] KDIGO clinical practice guidelines for acute kidney injury. *Kidney International Supplements*. 2012;2(1). doi:10.1038/kisup.2012.1.

[2] Makris K, Spanou L. Acute Kidney Injury: Definition, Pathophysiology and Clinical Phenotypes. *Clin Biochem Rev*. 2016;37(2):85-98.

The correct answer is: Hepatorenal syndrome

Question 9

ID: 38898

Incorrect

Flag question

Send Feedback

Which of the following scenarios would constitute an acute kidney injury (AKI) based on the current KDIGO guidelines?

Select one:

- DF, a 27-year-old female, has been hospitalized for 2 days and her serum creatinine increased from 120 umol/L to 190 umol/L due to sepsis ✔
- KB, a 31-year-old male, has been hospitalized for 24 hours with a kidney stone and his serum creatinine increased from 64 umol/L to 83 umol/L ✖
- An 80-year-old female's serum creatinine increased from 85 umol/L to 120 umol/L with a decrease in urine output over the past 7 days ✖

  Rose Wang (ID:113212) this answer is incorrect. The serum creatinine increase is concerning, but it is less than a 1.5 times increase from baseline over 7 days.

- FB, a 91-year-old female, has not had a change in serum creatinine but her urine output has decreased significantly in the past day ✖

Incorrect

Marks for this submission: 0.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To understand the definition of acute kidney injury (AKI).

BACKGROUND:

Acute kidney injury (AKI) occurs when there is a rapid decrease in kidney function. AKI can be classified based on where the damage or impairment is occurring in the kidney:

- *Pre-renal AKI* occurs when there is hypoperfusion of the kidney (the most common cause of AKI)
- *Intrinsic AKI* occurs when there is damage or impairment within the kidney
- *Post-renal AKI* occurs when a severe blockage beyond the kidney (e.g., in the ureter) causes waste buildup in the kidney

Possible causes of AKI

Impairment Location	Possible causes of AKI

Impairment Location	Possible causes of AKI
Pre-renal AKI	• Hypovolemia • Increased vascular resistance • Reduced cardiac function • Systemic vasodilation
Intrinsic AKI	• Bilateral renal artery stenosis • Infection • Immune system dysfunction (e.g., lupus, IgA glomerulonephritis) • Nephrotoxic drugs
Post-renal AKI	• Blood clots • Improperly placed catheter • Kidney stones (nephrolithiasis) • Urogenital cancers

AKI is defined as any of the following:

- Increase in SCr by ≥26.5 umol/L within 48 hours, or
- Increase in SCr by ≥1.5 times the baseline within 7 days, or
- Urine volume of <0.5 ml/kg/hour for 6 hours

In addition to reduced or lack of urine output, fluid retention is a very common sign of AKI. Nausea/vomiting can also occur as AKI results in a buildup of waste in the blood.

RATIONALE:

Correct Answer:

(Option #1): DF's serum creatinine has increased by more than 26.5 umol/L over 48 hours.

Incorrect Answers:

(Option #2): The guidelines state that an increase of at least 26.5 umol/L is required for the diagnosis of acute kidney injury (AKI).
(Option #3): The serum creatinine increase is concerning, but it is less than a 1.5 times increase from baseline over 7 days.
(Option #4): There is not enough information provided in this option to determine if FB is experiencing an acute kidney injury (AKI).

TAKEAWAY/KEY POINTS:

The KDIGO guidelines require a specific change in serum creatinine (SCr) over 2 to 7 days in order to identify acute kidney injury (AKI).

REFERENCES:

[1] KDIGO clinical practice guidelines for acute kidney injury. *Kidney International Supplements*. 2012;2(1). doi:10.1038/kisup.2012.1.

[2] Makris K, Spanou L. Acute Kidney Injury: Definition, Pathophysiology and Clinical Phenotypes. *Clin Biochem Rev*. 2016;37(2):85-98.

The correct answer is: DF, a 27-year-old female, has been hospitalized for 2 days and her serum creatinine increased from 120 umol/L to 190 umol/L due to sepsis

Question 10

ID: 46617

Correct

Flag question

Send Feedback

You are a clinical pharmacist who has taken on a preceptor role for an Advanced Pharmacy Practice Experience (APPE) student at a large teaching hospital. Today, your student approaches you for some education about medication-induced acute kidney injury (AKI). Her patient, DN, is a 62-year-old man who has recently been diagnosed with heart failure with reduced ejection fraction (HFrEF) by his cardiologist in the community. His cardiologist prescribed new medications for DN, including ramipril 2.5 mg BID, bisoprolol 1.25 mg daily, spironolactone 12.5 mg daily, dapagliflozin 10 mg daily, and furosemide 20 mg daily. His past medical history is significant for dyslipidemia and bipolar disorder for which he takes rosuvastatin 40 mg and lithium 600 mg TID, respectively. DN was hospitalized overnight due to progressive symptoms of vomiting and reduced urinary output over the past few days. The admitting physician suspects that he is experiencing an acute kidney injury (AKI) and has ordered tests. Your student would like to discuss with you which of DN's medications may have caused the AKI.

All of the following medications are known to cause acute kidney injury (AKI) **EXCEPT**:

Select one:

- Ramipril ✖
- Bisoprolol ✔

Rose Wang (ID:113212) this answer is correct.
Bisoprolol does not cause AKI.

- Furosemide ✖
- Lithium ✖

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To understand how common medications can negatively impact the kidneys.

BACKGROUND:

The incidence of acute kidney (AKI) in the community setting is very low compared to acute hospital settings. This is because hospitalized patients are more likely to develop AKI as patients are more prone to depleted plasma volumes (due to blood loss following surgeries, dehydration, and/or urinary catheter implantation) in these settings. Development of AKI is associated with increased morbidity; AKI can lead to longer hospitalizations, readmission, ventilator days, and need for post-hospitalization care. In the last 2 decades, the total number of AKI-related hospitalizations has increased 4-fold from 953,926 in 2000 to 3,959,560 in 2014. AKI tends to be more common in the critically ill hospitalized patient population (30-60%) compared to the non-critically ill hospitalized patients (3-18%).

Risk factors that increase risk of AKI include CKD, diabetes, cardiac or hepatic disease, albuminuria, major surgery (especially cardiac surgery), acute decompensated heart failure, sepsis, hypotension, fluid loss (diarrhea, vomiting, or dehydration), medications (angiotensin-converting enzyme [ACE] inhibitors, angiotensin receptor blockers [ARBs], lithium, aminoglycosides, etc.), advanced age, male gender, and African American race. Up to 60% of AKI cases are drug-induced.

70% of AKI cases are pre-renal AKI, which is due to decreased perfusion to the kidneys. They are typically caused by conditions that decrease cardiac output and perfusion (e.g., dehydration, severe blood loss, sepsis, severe burns, and cirrhosis), renal arterial disease, and medications (such as ACE inhibitors, diuretics, and NSAIDs). ACE inhibitors and ARBs inhibit angiotensin-II activity which normally would constrict the efferent arteriole when the perfusion pressure in the kidneys is low. If perfusion is low, filtration remains low and this results in poor kidney function. NSAIDs inhibit the production of prostaglandins which normally promotes vasodilation of the afferent arteriole when perfusion is low. Diuretics promote diuresis which leads to depleted blood volume and decreased perfusion.

Intrinsic renal failure makes up 20% of the AKI cases. Acute tubular necrosis (ATN) can be caused by ischemia (due to hypotension or sepsis), nephrotoxic substances (such as lithium, radiocontrast dye, aminoglycosides, cisplatin, carboplatin, cyclosporine, and tacrolimus). Not only is lithium a nephrotoxic medication, but it leads to desensitization of antidiuretic hormone (ADH). Desensitization of ADH leads to inability to concentrate urine and increased diuresis (this condition is known as diabetes insipidus). Acute interstitial nephritis (AIN) is typically caused by medications such as penicillin and nonsteroidal antiinflammatory drugs (NSAIDs). Glomerulonephritis is caused by glomerular diseases, autoimmune diseases (e.g., systemic lupus erythematosus (SLE)), and medications (e.g., lithium and NSAIDs).

Post renal typically occurs due to obstruction or hypertrophy of renal pelvis, ureters, and/or bladder. Obstructions include nephrolithiasis (kidney stones), nephrocalcinosis (calcium deposits along the urinary tract), and bladder outlet obstruction (improper urinary catheter placement leading to blockage of urinary tract). It can also be caused by malignancies, medications that cause urinary retention (e.g., anticholinergic medications), other medication (e.g., methotrexate, penicillin, and sulfamethoxazole trimethoprim (Septra)).

RATIONALE:

Correct Answer:

(Option #2): Bisoprolol does not cause AKI.

Incorrect Answers:

(Option #1): Ramipril can cause acute kidney injury (AKI) by inhibiting angiotensin II activity which normally would constrict the efferent arterioles when the perfusion pressure in the kidneys is low.
(Option #3): Diuretics promote diuresis which leads to depleted blood volume and decreased perfusion of the kidneys.
(Option #4): Lithium is a nephrotoxic drug and can also lead to desensitization of antidiuretic hormone (ADH), which leads to an inability to concentrate urine and increased diuresis.

TAKEAWAY/KEY POINTS:

NSAIDs can cause acute kidney injury (AKI) by inhibiting the production of prostaglandins which normally promote vasodilation of the afferent arterioles when perfusion is low.

REFERENCES:

[1] Flurie RW. Disorders of Potassium and Magnesium Homeostasis. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e*. McGraw-Hill.

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The correct answer is:
Bisoprolol

Finish review